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THE SIGNIFICANCE OF CARBON MONOXIDE IN THE URBAN ATMOSPHERE

The topic of effect of carbon monoxide on health has so far been the domaine of general medicine which has analysed it in substantial detail. The urban hygienist, on the other hand, so far has not been concerned with the problem to the extent required and, indeed, paid very little professional attention to it.

The increasing number of motor vehicles which, with the exception of heating plants, are the principal generators of carbon monoxide in the urban atmosphere, turned the attention of the urban hygienist to this problem as one of the major smog generators.

The Occurrence of Carbon Monoxide in the Atmosphere

The volume of carbon monoxide measured in "pure areas" is low and is generally stated to range between 0.01 and 0.2 mg/m^3 [17]. Values measured in the atmosphere over the North Pacific reached about 0.029 mg/m^3 [27], while values measured in unpopulated areas of North Carolina ranged between 0.6 and 1.2 mg/m^3 [28].

Accurate data on the chemical change of carbon monoxide in the atmosphere are not available. Certain studies suggest that CO may dissipate in low atmosphere within three to five years [27]. There are estimates which indicate that in the absence of decomposition or degradation of carbon monoxide in the atmosphere, the content would

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increase from 0.03 mg/m^3 to 1 mg/m^3 [3, 11, 27] in non-polluted areas, given the fact that industrial sources elsewhere generate 1.8 - 2.1 x 10^{11} kg annually.

Degradation of carbon monoxide occurs through (1) travel to higher atmosphere by oxidation to carbon dioxide; (2) metabolic change to carbon dioxide and methane with the aid of microorganisms in the soil; and (3) the binding process of porphyric components of plants and organisms and through the interaction of carbon monoxide and ocean water which changes it to carbon monoxide.

Figure 1. Daily fluctuation of carbon monoxide as measured at three locations in Detroit. Concentration values given in ppm [34]

In the United States it is believed that more than 50% of all carbon monoxide production in the atmosphere is generated by the automobile [6, 13, 30, 5]. The absolute value is estimated at 5.9 x 10^7 tons of CO annually. Air transportation accounts for a mere two per cent [37]. Other sources of carbon monoxide in the atmosphere are heating plants and households. In terms of percent, carbon monoxide is the heaviest component of all those entering the atmosphere.

The concentration of carbon monoxide in the urban atmosphere varies widely depending on location and time. In daytime, there is a high correlation between the concentration of carbon monoxide in the

atmosphere and automobile traffic [13, 24, 30, 36]. The correlation coefficient of automobile traffic and carbon monoxide in the atmosphere ranged between 0.75 and 0.95 in U.S. cities [24, 29, 35].

There are two peaks of CO concentration in the atmosphere, one in the morning and one in the afternoon, each relating to the amount of automobile traffic as indicated in Figure 1.

Over the span of a year, the highest values are recorded in the fall, followed by the summer, spring, and winter. This is caused by the relatively high atmospheric stability of the air in the fall and summer, and by lower wind velocity than is the case during the other two seasons.

Meteorological phenomena which influence the extent of CO concentration in the atmosphere are the following, in order of importance: wind velocity, wind direction, turbulence, and atmospheric stability. In addition to the macrometeorological factors, there are micrometeorological factors, such as air turbulence caused by the moving automobile, patterns of residential developments, and others. These factors are influential where wind velocity drops to less than 1.3 m/sec.

During unfavorable weather situation, such as temperature inversion, there may be a sharp increase in the concentration of carbon monoxide. For example, an eight-hour daily concentration of carbon monoxide of 35 mg/m^3 [32], was recorded in Los Angeles in 1964. A peak concentration of 270 mg/m^3 and 415 mg/m^3 was recorded in London in 1957 [18].

By comparing CO concentration in the open atmosphere and inside homes, it was found that concentration of CO in homes is more dependent on local (in-house) conditions than outside atmosphere.

Studies conducted in the US indicate that carbon monoxide levels would be 30% higher in 1985 than in 1964 if nothing is done about automobile exhaust systems.

The Effect of Carbon Monoxide on Plants and Organisms

Plants evidence a very low sensitivity to carbon monoxide in the atmosphere. Of all the components contained in the atmosphere, carbon monoxide has the least effect on the life of plants. Plants are by far more sensitive to other compounds, including ethylene, sulphur dioxide, and the like.

As indicated by published experiments, 100 kinds of plants were exposed to the effects of high concentration of carbon monoxide (from 115 to 575,000 mg/m^3). No damage to growth or any other changes were recorded under concentrations of 115 mg/m^3 for 7 to 23 days [33]. Under high concentrations, leaves began to twist, buds were damaged, and growth was retarded.

Carbon monoxide affects microorganisms by inhibiting the fixation of nitrogen [29]. The capability of fixating nitrogen compounds through microorganisms is essential to the development of plants; damage is caused by high concentration of carbon monoxide (2300 mg/m^3 for a period of 35 hours)[21].

In the case of macroorganisms the basic toxic property is the reaction of carbon monoxide with hemoproteins. The infinite constant

of carbon monoxide to hemoglobulin is by certain authors reported to be between 200 and 300. According to the most recent studies, the value of 200 is the most probable for human pathophysiology. The presence of carbon monoxide in blood causes a change in the dissociation curve of oxihemoglobulin, causing disruption in the deposit of oxygen in the fiber tissue. Both reactions, i.e. saturation of blood with carbon monoxide and with oxygen, depend on partial pressure of the gases in the environment.

In addition to adversely affecting hemoproteins, carbon monoxide has been proven to affect **cytochromoxidasis** by blocking its activity. Lilienthal states that in regard to the afinity of carbon monoxide to numerous respiratory pigments such as hems, oxydases, and catalases, selective effects of carbon monoxide may cause partial damage to specific systems via other mechanisms other than the commonly known anoxemy, caused by the formation of carbonyl hemoglobin; in addition, it is suspected that carbon monoxide may adversely affect a wide spectrum of tetrapyrolic respiratory pigments.

The human organism is constantly exposed to low concentrations of the so-called endogenous carbon monoxide created by catabolic reactions of hemoglobin. This quantity is stated to be on the order of 0,4 ml carbon monoxide per hour. Endogenous formation is understood to be 0,5 to 0,8% carbonyl hemoglobin under normal "background" conditions.

Many methods of determining carbonyl hemoglobin are known, particularly in medicine. These methods, however, are not sensitive enough to measure low levels of carbonyl hemoglobin. For purposes of urban hygiene, more accurate means of measurement are necessary to determine

levels also 3% COHb.

Noble methods can be divided into three basic groups:

1. methods which do not result in the destruction of substrate;
2. methods which result in the destruction of substrate;
3. balanced methods, producing neither result above.

1. Nondestructive methods include primarily spectrophotometric methods. The advantage lies in the low quantity of blood required, no necessity of establishing the quantity of hemoglobin, simplicity, and speed. However, values less than 3-4% COHb cannot be obtained reliably.

2. Destructive methods are based on the principle of releasing carbon monoxide from blood corpuscles and measuring its content. These methods require a larger quantity of blood, the level of hemoglobin must be simultaneously established, but the accuracy is high.

3. Balanced methods rely on the fact that the content of carbon monoxide in exhalation is equal to its content in the lungs, after breath has been withheld for 20 seconds.

The effect of carbon monoxide on human organism is related to (1) level of concentration; (2) time of exposure; and (3) quantity of passing air.

As evidenced by tests, a balanced condition ensues after a certain period of exposure. On the basis of these tests, standards have been developed which are sufficiently informative about the COHb values under various concentration levels of carbon monoxide in the

atmosphere. A simple formula for an eight-hour exposure is the following [16]:

COHb in % = [0,16 x (CO) in ppm*] + 0,5

0,5 background

When samples for a shorter period of time are required, the following formula can be used:

log % COHb = 0,85753 log CO in ppm + 0,62005 log t - 2,29519

t = exposure in minutes

Based on this equation, a nomograph has been developed [25] and is shown in Figure 2.

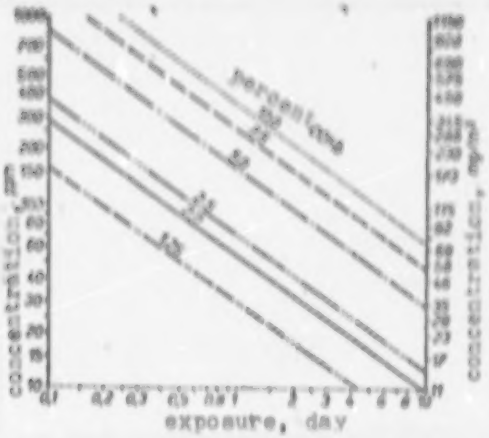

Figure 2. The relationship of concentration and exposure to COHb in percent [25].

Long term exposure of animals to carbon monoxide resulted in morphological changes in heart musculature and brain. Dogs exposed intermittently or continually to 58 mg/m^3 for a period of 6 weeks displayed abnormal EEG within three weeks [22]. An autopsy of the animals indicated mobilization of the glia and dilatation of lateral brain chambers. Another study established, after a three-week exposure, a significant increase in hemoglobin and hematocrit. Concentrations of 115 mg/m^3 for a period of six weeks produced dilatation of the

* 1 ppm = 0,001 vol. % = 1.2 mg/m^3

right heart chamber along with degenerative changes of heart muscle tature [22].

Study of carbon monoxide effect on the blood system of animals suggests the possibility of adaptation to carbon monoxide. Rats exposed to 100 mg/m^3 CO for eight weeks exhibited an increased level of hemoglobin after the first five weeks, without a further increase in the following three weeks. When the concentration was increased to 300 mg/m^3, a further increase in hemoglobin was discovered [3, 26].

Short-term tests with a concentration of 58 mg/m^3 for one hour on rats produced changes in the EEG. A return to normal was observed 48 hours after exposure [31].

Controlled long-term, low concentration tests on humans have not been conducted. The only observations available are those from the area of medicine.

Controlled short-term tests indicate that the central nervous system reacts in cases where COHb values exceed 2%; for example, exposure to 58 - 200 mg/m^3 for a period of two hours indicated a significantly reduced ability to tell time [3]. Another experiment unveiled impaired visual functions. Psychomotoric ability was impaired when patients were exposed to concentrations about 5% COHb.

The effect of carbon monoxide on the cardiovascular system appeared both in oxidative metabolism and in veins, primarily myocardial. At values over 5% COHb, there was a relative reduction in partial pressure of oxygen in veins by 13.3% and in arteries by 7.3%. These changes correlated with increased ventilation and heart activity [2].

Changes in the metabolism of the cardiovascular system were observed in persons with reduced oxygenation in the system such as resulting from coronary and the like. A group of such patients was exposed to an atmosphere where the level of CO2% was about 1%. Patients in the control group reacted with a compensatory flow of blood through the coronary system, reflecting need for greater quantities of oxygen. This change was not evident in patients with coronary diseases. These and other results indicate that persons with cardiovascular problems are very sensitive to increased levels of carboxyl hemoglobin over 5%.

Another important factor is the fact that carbon monoxide reduces the partial pressure of oxygen, necessitating an increase in heart activity; an increase from 1% COHb to 13% COHb produces the same effect as atmospheric pressure difference between 100 m and 1900 m above sea level.

In summary, 0 - 1% COHb produces no changes; the physiological formation of endogenous oxygen ranges within these limits. At values 2 - 5% COHb, there is a reduction in visual activity, relative visual threshold, and impairment in psychomotor tests.

Values above 5% COHb produce increased cardiovascular activity, changes in the arteriovenose difference of oxygen, changes in oxygen supply of the cardiovascular system, and the like.

Epidemiological studies, which deal with the relationship of atmosphere polluted with carbon monoxide to population living in the affected area or the relationship between smoking and its effect on the organism, also evidenced the harmful effects of carbon monoxide.

Persons working in automotive garages, on traffic arteries, and the like, manifest the so-called syndrome of chronic poisoning by carbon monoxide, displaying headache, dizziness, breathing difficulties, diarrhea, frequent urinea, perspiring, thirst, loss of weight, low libido, reduced ability to smell, and insomnia.

An examination of 331 policemen in Paris, exposed to automobile traffic for 5 hours in concentrations 12 - 14 mg/m^3 CO, revealed no increase in the level of COHb in cigarette smokers, but significant increase in nonsmokers [15]. The concentration of COHb in medium heavy smokers is in the area of 5%. This is understandable when we realize that cigarette smoke contains more than 22400 mg/m^3 CO and that the average concentration inhaled is 460 - 575 mg/m^3 CO. Studies in mortality and morbidity indicate a very close relationship between mortality, worsening of heart or lung condition, and carbon monoxide level.

Increased levels of monoxide particularly affect persons who (1) have anemia with loss of sufficient saturation of tissue by oxygen; (2) have cardiovascular problems leading to circulatory damages; (3) have metabolic damages, such as tyreotoxicosis; (4) have a lung disease; and (5) fetus which is highly sensitive to lack of oxygen.

The above survey indicates that the problem of carbon monoxide is very serious and must be paid increased attention by the urban hygienist.

Summary: The author gives a brief review on the problematics of low concentrations of carbon monooxide in the open air and its influence on human organism. The main sources of carbon monooxide, its transformations in atmosphere and the dependence of its concentration on meteorological factors are described. The remarkable part of the review is devoted to the toxic effects of low levels of carbon monooxide on human and animal organism in controlled experimental conditions and in epidemiological studies. **Bs.**

BIBLIOGRAPHY

1. Astrup, P., Kjeldsen, K., Wanstrup, J., Effects of carbon monoxide on the arterial walls. Paper presented at the New York Academy of Sciences Conference on Biological Effects of Carbon Monoxide. New York City, January 12-14, 1970.

2. Ayres, S. M., et al., "Systematic and myocardial hemodynamic responses to relatively small concentrations of carboxyhemoglobin (COHb)," Arch. environm. Hlth., 18, 1969, pp. 699-709.

3. Beard, R. R., Wertheim, G. A., "Behavioral impairment associated with small doses of carbon monoxide," Amer. J. public Hlth, 57, 1967, pp. 2012-2011.

4. Berka, J., Kadlec, K., "Carbon Monoxide (source, use, methods of establishing levels, hygienic profile of working stations, physiological effects, clinic of acute and chronic poisoning)," Prague, SZdN, 1956.

5. Caplan, J. D., Causes and control of automotive emissions, Presented at the Institution of Mechanical Engineers, London, April 9, 1963.

6. Elliot, M. A., Nebel, G. J., Rounds, F. G., "The composition of exhaust gases from diesel, gasoline and propane powered motor coaches," J. Air Polt. Control Ass., 5, 1955, 2 : 103-108.

7. McFarland, R. A., et al., "The effects of carbon monoxide and altitude on visual thresholds," Aviation Med., 15, 1944, pp. 381-394.

8. Goldsmith, J. B., Schuette, F., Novick, L., "Appraisal of carbon monoxide exposure from analysis of expired air," Exc. med. Int. Congr. Series 62, 1963, pp. 948-952.

9. Goldsmith, J. B., Terzaghi, J., Hackney, J. D., "Evaluation of fluctuating CO exposures," Arch. environm. Hlth, 7, 1963, pp. 647-663.

10. Haagen-Smith, A. J., "Carbon monoxide levels in city driving," Arch. environm. Hlth, 12, 1966, pp. 548-550.

11. Haagen-Smith, A. J., Wayne, L. G., "Atmospheric reactions and scavenging processes," In: Air Pollution, Stern, A. C. (ed.) Vol. I, 2nd ed., New York, Academic Press 1968, pp. 149-186.

12. Haddon, W., Jr., Nesbitt, R. E., Garcia, R., "Smoking and pregnancy: Carbon monoxide in blood during gestation and at term," Obstet. Gynecology, 18, 1961, 3, pp. 262-267.

13. Hagen, D. F., Holiday, G. W., "The effects of engine operation and design variables on exhaust emissions" (SAE Paper No. 486 C) In: Vehicle emissions, Vol. 6, New York, Society of Automotive Engineers, Inc., 1964, pp. 206-223.

14. Hechter, H. H., Goldsmith, J. R., "Air pollution and daily mortality," Amer. J. Med. Sci., 241, 1961, pp. 581-588.

15. Chovin, P., "Carbon monoxide: Analysis of exhaust gas investigations in Paris," Environm. Res., 1, 1967, 2, pp. 198-216.

16. McIlvaine, P. M., Nelson, W. C., Bartlett, D., Jr., "Temporal variation of carboxyhemoglobin concentrations," Arch. environm. Hlth 19, 1969, pp. 83-91.

17. Junge, C. E., Air chemistry and radioactivity, New York Academic Press, 1963, p. 382.

18. Lawther, P. J., Commins, B. T., Henderson, M., "Carbon monoxide in town air," An interim report. Ann. acc. Hyg., 1962, 5, pp. 241-248.

19. Lewey, F. H., Drabkin, D. L., "Experimental chronic carbon monoxide poisoning of dogs," Amer. J. Med. Sci. 208, 1944, 4, pp. 502-511.

20. Lilienthal, J. R., Jr., J. Pharmacol. exp. therap., 99, 1950, p. 324.

21. Lind, C. J., Wilson, P. W., "Carbon monoxide inhibition of nitrogen fixation by azotobacter," Arch. biochem. 1, 1942, 1, pp. 59-72.

22. Lindenberg, R., et al., "An experimental investigation in animals of the functional and morphological changes from single and repeated exposure to carbon monoxide," Presented at the American Industrial Hygiene Conference. Washingon, D. C. May 13-17, 1962.

23. Navratsl, M., Prac. Lek., 7, 1955, 26.

24. Ozolins, G., Smith, R., "A rapid survey technique for estimating community," Air Pollution Emissions, National Air Pollution Control Administration, Cincinnati, Ohio. PHS Publication Number 999-AP-29, 1967, p. 77.

25. Peterson, J. E., Stewart, R. D., "Absorption and elimination of carbon monoxide by inactive young men," Report prepared under contract CRC-APRAC. Project No. CAPM-3-68. Coordinating Research Council, Inc. 1969.

26. Prezioni, T., "An experimental investigation in animals of the functional and morphologic effects of single and repeated exposures to high and low concentrations of CO," Paper presented

at the New York Academy of Sciences conference on biological effects of carbon monoxide. New York City, 1970, January 12-14.

27. Robbins, R. C., Borg, K. M., Robinson, E., "Carbon monoxide in the atmosphere," J. Air Pollut. Control Ass., 1968, 18, pp. 106-110.

28. Robinson, E., Robbins, R. C., "Atmospheric CO concentrations on the Greenland Ice Cap," Jour. Geophysical Research 74 (8), 1968-1973, April 15, 1969.

29. Rose, A. H., et al., "Comparison of auto exhaust emissions in two major cities," J. Air Pollut. Control Ass., 1965, 15, pp. 362-366.

30. Rose, A. H. Jr., "Summary report of vehicular emissions and their control," Paper presented at the St. Louis Section meeting of the society of automotive engineers. St. Louis, Mo., January 1969.

31. Rousel, A., Stupfel, M., Bouley, G., "Trends of experimental research on air pollution in France," Arch. environm. Hlth., 18, 1969, pp. 613-622.

32. Tebbens, B. D., "Gaseous Pollutants in the Air," In: Air Pollution, Stern A. C. (ed), Vol. 1, 2nd ed. New York Academic Press 1968, pp. 28-33.

33. Zimmerman, P. W., Crocker, W., Hitchcock, A. E., "Initiation and stimulation of roots from exposure of plants to carbon monoxide gas contrib," Boyce Thompson Inst. 5, 1933, 1, pp. 1-17.

34. Air Quality Criteria for Carbon Monoxide. National Air Pollution Control Administration Publication No. AP-62. March 1970.

35. Motor Vehicle Pollution in California. California Dept. of Public Health, Los Angeles, January 1967.

36. National Air Pollution Control Administration, Reference Book of Nationwide Emissions. U. S. DHEW, PHS, CPEHS, NAPCA, Durham, N. C.

37. Nature and Control of Aircraft Engine Exhaust Emissions Report of the Secretary of Health, Education, and Welfare to the United States Congress. Pursuant to Public Law 90-148, the Air Quality Act of 1967. U. S. Dept. of Health, Education and Welfare, Washington, D. C. U. S. Government Printing Office, Senate Document No. 91-9, 91st Congress. 1st Session. March 4, 1969, p. 32.

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